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Original Article

Study on Treatment and Outcome of Patients with Guillain–Barre Syndrome – A Prospective Study

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Abstract

Introduction: The history of Guillain–Barre syndrome (GBS) runs parallel with the discovery of the peripheral nervous system. Up to the second half of the 19th century, injury to the peripheral nervous system had not yet emerged as a possible cause of palsy.

Aim: This study aims to study the treatment and outcome in patients with various subtypes of GBS.

Materials and Methods: Patients who had been admitted with the diagnosis of GBS based on Asbury's criteria which included ascending areflexic quadriparesis, with or without cranial nerve dysfunction, and evolving within a period of 4 weeks. We also included patients who presented with features of GBS subtypes without prominent weakness. A detailed history and physical examination as per a structured pro forma were taken and necessary laboratory investigations were done.

Results: Twenty-five patients were admitted with disability grade of >3 in GBS disability scale (>5 in Medical Research Council disability scale) and 18 were admitted below the score. Sixteen patients needed ventilator support and 10 patients expired. Among the 22 patients who were treated with intravenous immunoglobulin (IvIg), 14 patients (24.6%) had a good outcome and 8 patients (14.0%) had a poor outcome. Among the nine patients who were treated with plasma exchange, six patients had a good outcome and three had a poor outcome. Among the 19 patients, who were treated with injection methylprednisolone, eight patients had a good outcome and 11 patients had a poor outcome. The values obtained are not statistically significant ($P = 0.076$).

Conclusion: The mean improvement in GBS disability scale from admission to the end of the 8th week is more for IvIg-treated patients when compared to methylprednisolone-treated group.

Key words: Guillain–Barre syndrome, Life threatening, Outcome

INTRODUCTION

Guillain–Barre syndrome (GBS) is an acute, self-limited, inflammatory, autoimmune disorder of the peripheral nervous system triggered usually by a bacterial or viral infection or other antecedent events.[1] It affects 0.9–2/100,000 persons in a year, with a worldwide distribution and a slight male preponderance.[2] In general, at the end of 1 year of illness, 5% of the patients had expired and 15% might be unable to walk. Hence, it causes a large loss of productivity and burdens health care due to its prolonged morbidity. It is a heterogeneous disorder in its type, severity, pathogenesis, and prognosis. GBS is characterized by rapidly progressive weakness of all four limbs with or without sensory loss, evolving within 4 weeks followed later by slow clinical and electrophysiological recovery.[3,4] The subtypes of GBS are several. Among those who produce weakness, the common one is acute inflammatory demyelinating polyradiculopathy (AIDP), acute motor-sensory axonal neuropathy (AMSAN), and acute motor axonal neuropathy (AMAN) and the rare one are pharyngo-cervico-brachial variant, bilateral foot drop, and bifacial weakness. Among those who do not produce weakness, the common one is Miller-Fisher syndrome (MFS) and the rare ones are pure sensory variant and acral paresthesias with areflexia.[1]

Neurophysiologic abnormalities are often very mild or occasionally normal in the early stages of GBS and hence may not correlate well with clinical disability. AIDP is

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characterized classically by conduction block with also prolongation of compound muscle action potential (CMAP) latency and F-wave latency but a normal amplitude. AMAN and AMSAN are characterized by reduction or absence of amplitude of CMAP and both CMAP and sensory nerve action potential, respectively.[5]

Experimental evidence implicates autoantibodies to gangliosides as the cause of the axonal subgroup of GBS and MFS. These antibodies may be generated by the immune response to an infective organism such as *Campylobacter jejuni*, cross-reacting with the epitopes on the axon. The resemblance of AIDP to experimental autoimmune neuritis suggests pathogenetic mechanisms involving T-cell induced macrophage-associated demyelination. This proposed autoimmune etiology leads to the induction of immunotherapy.[6]

Aim

This study aims to study the treatment and outcome in patients with various subtypes of GBS.

MATERIALS AND METHODS

This is a prospective study, conducted in patients who had been admitted with the diagnosis of GBS, in the medical, neuromedical, emergency medical, or intensive medical care ward in a tertiary care hospital.

Table 1: Severity of disability with which patient presented

GBS	Frequency	<3 – GBS score <5 – MRC disability scale [19] (30.2%)	>3 – GBS score >5 – MRC disability scale [38] (60.3%)
AIDP	43	18	25
AMSAN	8	-	8
AMAN	3	1	2
Unclassified	3	-	3

GBS: Guillain–Barre syndrome, MRC: Medical Research Council, AIDP: Acute inflammatory demyelinating polyradiculopathy, AMSAN: Acute motor-sensory axonal neuropathy, AMAN: Acute motor axonal neuropathy

The inclusion criteria consist of patients who presented with features of GBS based on Asbury's criteria which included ascending areflexic quadriparesis, with or without cranial nerve dysfunction, and evolving within a period of 4 weeks. We also included patients who presented with features of GBS subtypes without prominent weakness.

The exclusion criteria are as follows:

1. Early and prominent bladder and bowel dysfunction
2. Marked and persistent asymmetry of symptoms and signs
3. Presence of persistent sharp sensory level
4. Features of other diseases such as myasthenia gravis, botulism, poliomyelitis, porphyria, and diphtheria
5. Drug or toxin-induced acute neuropathy.

Data regarding the demographic features such as age, sex distribution, and month of occurrence and clinical features such as antecedent illness, the involvement of cranial nerves, and autonomic dysfunction were collected.

During admission, patients were analyzed for their disability using the GBS disability scale and Medical Research Council (MRC) disability scale. For patients with disability grade of >3 in GBS disability scale and for those with progressively increasing weakness, the definite treatment options (intravenous immunoglobulin [IvIg] or plasma exchange) were started. Due to non-availability, some patients received only injection methylprednisolone.

Patients were followed up throughout their stay in the hospital. Intensive medical care was provided for those patients with an advanced stage of the disease. Elective intubation was done for those patients who had poor single breath count estimation and reduced peak expiratory flow rate and for those with neck muscle weakness and poor cough reflex. Ventilatory support was provided for those in need. Tracheostomy was performed on those patients who tend to require ventilatory support for more than 10–14 days.

Periodic assessment of their clinical status and disability was done and their peak disability was noted. At the end of

Table 2: Treatments given for various subtypes of GBS

GBS	IvIg [22] (34.9%)	Methylprednisolone [25] (39.7%)	Plasma exchange [9] (14.3%)	No. treatment [4] (6.3%)	Not applicable [3] (4.8%)
AIDP [43]	18	15	6	4	-
AMSAN [8]	3	3	2	-	-
AMAN [3]	1	1	1	-	-
MFS [5]	-	5	-	-	-
Pure sens [1]	-	1	-	-	-
Unclassified [3]	-	-	-	-	3

GBS: Guillain–Barre syndrome, IvIg: Intravenous immunoglobulin, AIDP: Acute inflammatory demyelinating polyradiculopathy, AMSAN: Acute motor-sensory axonal neuropathy, AMAN: Acute motor axonal neuropathy, MFS: Miller–Fisher syndrome

8 weeks duration, reassessment was done in their clinical status and prevailing disability score was noted for further analysis.

RESULTS

Of the 63 patients, 20 patients were in the age group below 20 years and 32 patients were in the age group between 20 and 40 years. Regarding the sex distribution, there is a slight male preponderance of 35 males. Twenty-nine patients in the study had a history of antecedent illness preceded by the occurrence of GBS. Upper respiratory infection and diarrhea were noted in each of 13 patients.

In the present study, 25 patients were admitted with disability grade of >3 in GBS disability scale (>5 in MRC disability scale) and 18 were admitted below the score. The score is applicable for the GBS subtypes which cause weakness Table 1.

Treatments Given for Subtypes

In our study, IvIg was given to 18 patients, plasma exchange was given for nine patients, and injection methylprednisolone was given for 25 patients. No specific treatment was provided for four patients who presented with a very minimal disability and they improved spontaneously. Three patients in the study presented in a very acute form, with severe disability scores and died before any specific form of treatment were initiated Table 2.

Subtypes of GBS Patients Who Needed Ventilator or Ended Up in Death

In the present study, 16 patients needed ventilator support and 10 patients expired. Both the need for ventilator support and occurrence of death are noted in AIDP and AMSAN group and in those patients who presented with the very severe form of illness (a fulminant form of illness) Table 3.

Among the 63 patients, death was the outcome for 10 patients.

Twenty patients in our study needed ventilator support at some time during the course of hospital stay. Conventionally, considered poor prognostic factors not only influence death but also lead to a poor respiratory function requiring ventilatory support Tables 4 and 5.

Table 3: Subtypes of GBS patients who needed ventilator or ended up in death

GBS	Ventilator [20] (31.7%)	Death [10] (15.9%)
AIDP	13	5
AMSAN	4	2
AMAN	-	-
MFS	-	-
Pure sens	-	-
Unclassified	3	3

GBS: Guillain–Barre syndrome, AIDP: Acute inflammatory demyelinating polyradiculopathy, AMSAN: Acute motor-sensory axonal neuropathy, AMAN: Acute motor axonal neuropathy, MFS: Miller–Fisher syndrome

Table 4: The poor prognosticators associated with the patients who had expired

GBS	%	Ventilator [10]	Autonomic disturbance [9]	Peak of weakness ≤8 days [10]	Bulbar dysfunction [9]	Elderly population [2]	High-grade disability* at presentation [9]	Diarrhea [4]
AIDP [5]	11.6	5	4	5	3	2	4	2
AMSAN [2]	25	2	2	2	1	-	2	1
AMAN [0]	-	-	-	-	-	-	-	-
MFS [0]	-	-	-	-	-	-	-	-
Pure sens [0]	-	-	-	-	-	-	-	-
Unclassified [3]	100	3	3	3	3	-	1	1

*>3 scores in GBS disability grading; >5 scores in MRC disability grading. AIDP: Acute inflammatory demyelinating polyradiculopathy, AMSAN: Acute motor-sensory axonal neuropathy, AMAN: Acute motor axonal neuropathy, MFS: Miller–Fisher syndrome

Table 5: The poor prognosticators associated with the patients who required ventilatory support

GBS	%	Death [10]	Autonomic disturbance [13]	Bulbar dysfunction [9]	Peak disability reached<8 days [16]	Elderly [2]	High-grade disability at presentation* [19]	Diarrhea [7]	Tracheostomy [5]
AIDP [13]	30	4	8	5	10	2	12	4	5
AMSAN [4]	50	2	3	1	3	-	4	2	0
AMAN	-	-	-	-	-	-	-	-	-
MFS	-	-	-	-	-	-	-	-	-
Pure sens	-	-	-	-	-	-	-	-	-
Unclassified [3]	10	3	2	3	3	-	1	1	0

*>3 scores in GBS disability grading; >5 scores in MRC disability grading. AIDP: Acute inflammatory demyelinating polyradiculopathy, AMSAN: Acute motor-sensory axonal neuropathy, AMAN: Acute motor axonal neuropathy, MFS: Miller–Fisher syndrome

Table 6: Outcome related events for patients with poor prognostic factors

Prognostic factors	Death	Ventilator dependence	Tracheostomy	Poor outcome*	Good outcome**
Bulbar±Facial weakness, *P*=0.002#					
AIDP [21]	4	10	4	11	10
AMSAN [2]	1	1	-	2	-
AMAN [1]	1	-	-	1	-
Unclassified [3]	3	3	-	3	-
Autonomic dysfunction, *P*=0.029#					
AIDP [17]	4	6	2	8	9
AMSAN [5]	2	2	-	3	2
AMAN [1]	-	-	-	1	0
Unclassified [2]	2	2	-	2	0
Diarrhea, *P*=0.165#					
AIDP [8]	2	4	3	6	2
AMSAN [2]	1	2	-	2	-
AMAN [2]	-	-	-	-	2
Unclassified [1]	1	1	-	1	-
Peak disability reached within 8 days, *P*=0.044#					
AIDP [22]	5	8	2	11	11
AMSAN [6]	2	4	-	3	3
AMAN [3]	-	-	-	2	1
Unclassified [3]	3	3	-	3	-
Presented with a severe form of disability, *P*=0.001#					
AIDP [24]	4	12	5	12	12
AMSAN [8]	2	4	-	4	4
AMAN [2]	-	-	-	1	1
Unclassified [3]	3	3	-	3	-

*>3 scores in GBS disability grading; **≤3 scores in GBS disability grading; >5 scores in MRC disability grading; ≤5 scores in MRC disability grading; #Significance of influence of prognostic factors for poor outcome. GBS: Guillain–Barre syndrome, MRC: Medical Research Council, AIDP: Acute inflammatory demyelinating polyradiculopathy, AMSAN: Acute motor-sensory axonal neuropathy, AMAN: Acute motor axonal neuropathy

Table 7: Treatment adopted in GBS subtypes

GBS	IvIg	Methylprednisolone	Plasma exchange	Not applicable*	No treatment
AIDP					
Mild disability presentation [18]	6	6	2	-	4
Severe disability presentation [25]	12	9	4	-	-
AMSAN					
Mild disability [0]	-	-	-	-	-
Severe disable [8]	3	3	2	-	-
AMAN					
Mild disability [1]	-	1	-	-	-
Sever disability [2]	1	-	1	-	-
Unclassified					
Mild disability [0]	-	-	-	-	-
Severe disability [3]	-	-	-	3	-

*Not applicable – Patients died on day 1 or 2 before any form of treatments are effective. GBS: Guillain–Barre syndrome, MRC: Medical Research Council, AIDP: Acute inflammatory demyelinating polyradiculopathy, AMSAN: Acute motor-sensory axonal neuropathy, AMAN: Acute motor axonal neuropathy, IvIg: Intravenous immunoglobulin

In the study, the outcome assessment is based on whether the patients attained independent walking which is a good outcome (≤3 in GBS disability score and ≤5 in MRC disability scale) or did not attain independent walking which is a poor outcome, at the end of 8 weeks.

Among the 18 AIDP patients who were treated, six were from mild disability group and 12 were from severe disability group. Among the 15 AIDP patients who were treated with injection methylprednisolone, nine were from severe disability and six were from mild disability group.

Three AMSAN patients of severe disability were treated with IvIg and methylprednisolone and two by plasma exchange. All patients in the AMSAN group had presented with a severe disability.

One AMAN patient of severe disability group was treated each with IvIg and plasma exchange and one with a mild disability was treated with methylprednisolone Tables 6 and 7.

In the group of three patients who presented with the very acute and severe form of illness, no effective treatment was started before they expired.

Table 8: Comparison of treatment adopted with clinical outcome at 8 weeks among GBS subtypes

GBS	Death [7]	Ventilator support [16]	Tracheostomy [5]	Poor outcome [21]	Good outcome [30]
IvIg [22] *P*=0.410#					
AIDP [18]	1	5	1	6	12
AMSAN [3]	-	-	-	1	2
AMAN [1]	-	-	-	1	-
Plasma exchange [9] *P*=0.687#					
AIDP [6]	1	1	-	2	5
AMSAN [2]	1	2	-	1	1
AMAN [1]	-	-	-	-	1
Methylprednisolone [18] *P*=0.134#					
AIDP [15]	3	7	4	7	8
AMSAN [3]	1	1	-	3	-
AMAN (1)	-	-	-	-	1

Significance of influence of treatment options for the outcome. GBS: Guillain–Barre syndrome, AIDP: Acute inflammatory demyelinating polyradiculopathy, AMSAN: Acute motor-sensory axonal neuropathy, AMAN: Acute motor axonal neuropathy

Table 9: Comparison of treatment options with clinical outcome at 8 weeks among GBS subtypes who presented with severe disability

GBS	Death	Ventilator	Tracheostomy	Poor outcome	Good outcome
IvIg					
AIDP [12]	-	4	1	5	7
AMSAN	-	1	-	1	2
AMAN	-	-	-	1	-
Plasma exchange					
AIDP [4]	1	1	-	2	2
AMSAN [2]	1	1	-	1	1
AMAN [1]	-	-	-	-	1
Methylprednisolone					
AIDP [9]	3	6	3	6	3
AMSAN [3]	1	1	-	3	-
AMAN [0]	-	-	-	-	-

GBS: Guillain–Barre syndrome, AIDP: Acute inflammatory demyelinating polyradiculopathy, AMSAN: Acute motor-sensory axonal neuropathy, AMAN: Acute motor axonal neuropathy

Table 10: Mean improvement in disability scores for various treatment options

Disability scale	Treatment option	Total numbers	Mean improvement
GBS disability scale	IvIg	22	1.09
P=0.002#	Plasma exchange	9	0.67
	Methylprednisolone	19	0.00
	No treatment	4	1.00
	Not applicable	3	−1.00
MRC disability scale	IvIg	22	2.50
P=0.000#	Plasma exchange	9	1.33
	Methylprednisolone	19	0.42
	No treatment	4	2.00
	Not applicable	3	−1.67

#Significance of influence of treatment options on mean improvement in disability scores. GBS: Guillain–Barre syndrome, IvIg: Intravenous immunoglobulin, MRC: Medical Research Council

In the IvIg-treated AIDP group of 18 patients, 12 patients had a good outcome and 6 had a poor outcome. In the plasma exchange-treated AMSAN group of two patients, one each had a good and poor outcome. In the methylprednisolone-treated AIDP group of 15 patients, seven had a poor and eight had a good outcome, whereas three patients expired and 7 had ventilator support.

In the IvIg-treated AIDP patients (12) who presented with a severe disability, the outcome was poor in five patients and good in seven patients. Ventilator was needed in four patients and one had prolonged ventilatory support.

In the plasma exchange-treated (four) AIDP patients who presented with severe disability, two had poor and two had a good outcome. One patient had expired in this group after ventilator support.

In the methylprednisolone-treated (nine) AIDP patients who presented with a severe disability, the outcome was good for three and poor for six patients Tables 8-10.

DISCUSSION

In our study, IvIg was administered to 22 patients (34.9%), plasma exchange was given to 9 patients (14.3%), and injection methylprednisolone was given to 25 patients (39.6%). As already noted, three patients with a fulminant

form of illness were not able to receive either of these treatment modalities and were also not grouped in any of the GBS subtypes (unclassified in our study).

A total of 19 patients needed ventilatory support which constituted 31.7% of the total admission. In various studies, a similar incidence is noted.[7,8] Lawn *et al.*[9] noted that 53% of patients needed ventilatory support, whereas Kalitha *et al.*[10] noted only in 10% of patients. In the AIDP subtype, 27.9% of patients needed ventilatory support, whereas in the AMSAN type, the need was for 50% of cases.

A total of 10 deaths are recorded in our study which constituted 15.9% of the total admission. Even in well-equipped centers with aggressive intensive care unit care, the mortality is noted to be around 5–10%[6,7] and 4–15%.[11,12]

A total of five patients required tracheostomy for the need for prolonged ventilation which constituted 7.9% of the total admission.

The IvIg-treated AIDP patients clearly showed less percentage of death, need for ventilatory support and tracheostomy when compared with methylprednisolone-treated patients, though the values are statistically not significant. The other subtypes and variables are very small and hence cannot be compared.

Among the AIDP patients, who presented with a severe disability at admission,[13] 12 were treated with IvIg, four with plasma exchange, and nine with methylprednisolone.

The outcome was good for 7 patients (58.3%) and poor for 5 patients (41.6%) in the IvIg-treated group. The outcome was good for two patients and poor for two patients in the plasma exchange-treated group. The outcome was good for 3 patients (33%) and poor for 6 patients (66%).

Among the AMSAN patients, who presented with a severe disability at admission, three each were treated with IvIg and methylprednisolone, and two patients were treated with plasma exchange. The outcome was good for two and poor for one patient, treated with IvIg, whereas all three patients had a poor outcome in methylprednisolone-treated group. Plasma exchange resulted in one good and one poor outcome.

Even in the group of patients who presented with severe disability (>3 in GBS disability score) at admission, among the AIDP patients, the outcome appears to be better for those treated with IvIg compared to methylprednisolone. Among the AMSAN patients also, the outcome is poor for the methylprednisolone treated three patients. The values are statistically not significant.

Among the 12 AIDP patients, who presented with a severe disability at admission and treated with IvIg, one of 12 required tracheostomy (8%) and there was no death in this group.

Among the nine AIDP patients, who presented with a severe disability at admission and treated with injection methylprednisolone, six of nine required tracheostomy (33%) and there were three deaths in this group (33%).

Among the AIDP patients who presented with a severe disability at admission itself, the need for ventilatory support with or without tracheostomy and the death is more in methylprednisolone-treated group than in the IvIg-treated group.

The mean improvement in GBS disability scores from admission to the end of the 8th week was calculated for each treatment modality. For IvIg, it is 1.09; for plasma exchange, it is 0.67; and for methylprednisolone, it is 0.00. The values are statistically significant ($P = 0.02$).

Similarly, the mean improvement in MRC disability scores from admission to the end of the 8th week was calculated. For IvIg, it is 2.05; for plasma exchange, it is 1.33; and for methylprednisolone, it is 0.42. The values are statistically significant ($P = 0.00$).

For AIDP subtype of GBS patients, the mean improvement in GBS disability scores from admission to the end of the 8th week was calculated. For IvIg, it is 1.06; for plasma exchange, it is 0.67; and for methylprednisolone, it is 0.13. The values are statistically not significant.

Similarly, for AIDP patients, the mean improvement in MRC disability scores from admission to the end of the 8th week was calculated. For IvIg, it is 2.39; for plasma exchange, it is 1.33; and for methylprednisolone, it is 0.53. The values are statistically significant ($P = 0.0$).

CONCLUSION

A significant proportion of patients presents with peak disability within 8 days of onset of illness to whom definite treatment options are to be made available to enhance a good and early recovery because this is the group associated with poor outcome. The mean improvement in GBS disability scale from admission to the end of the 8th week is more for IvIg-treated patients when compared to methyl prednisolone-treated group, which is statistically significant. It is also applied well to the AIDP subtype of GBS. Although statistically not significant in this study, injection methylprednisolone is associated with a high percentage of the poor outcome when compared to IvIg

and plasma exchange. The prolonged morbidity of the illness evidenced by the need for tracheostomy is more for those treated with methylprednisolone when compared to other definite treatment options.

REFERENCES

1. Nguyen T, Taylor R. Guillain Barre Syndrome; 2019. Available from: https://www.ncbi.nlm.nih.gov/books/NBK532254. [Last cited on 2019 Jul 31].
2. Durcan FJ, Corbett JJ, Wall M. The incidence of pseudotumor cerebri. Population studies in Iowa and Louisiana. Arch Neurol 1988;45:875-7.
3. Asbury AK, Amason BG, Karp HR, McFarlin DF. Criteria for diagnosis of Guillain-Barre syndrome. Ann Neuro1 1998;3:565-6.
4. Asbury AK, Cornblath DR. Assessment of current diagnostic criteria for Guillain-Barré syndrome. Ann Neurol 1990;27:S21-4.
5. Griffin JW, Li CY, Ho TW, Xue P, Macko C, Gao CY, *et al.* Guillain-Barré syndrome in Northern China. The spectrum of neuropathological changes in clinically defined cases. Brain 1995;118:577-95.
6. Kaida K, Ariga T, Yu RK. Antiganglioside antibodies and their pathophysiological effects on Guillain-Barré syndrome and related disorders a review. Glycobiology 2009;19:676-92.
7. Goh KJ, Ng WK, Vaithialingam M, Tan CT. A clinical and electrophysiological study of Guillain Barre syndrome in Malaysia. Neurol J Southeast Asia 1999;4:67-72.
8. Durand MC, Porcher R, Orlikowski D, Aboab J, Devaux C, Clair B, *et al.* Clinical and electrophysiological predictors of respiratory failure in Guillain-Barré syndrome: A prospective study. Lancet Neurol 2006;5:1021-8.
9. Lawn ND, Fletcher DD, Henderson RD, Wolter TD, Wijdicks EF. Anticipating mechanical ventilation in Guillain-Barré syndrome. Arch Neurol 2001;58:893-8.
10. Kalita J, Misra UK, Das M. Neurophysiological criteria in the diagnosis of different clinical types of Guillain-Barre syndrome. J Neurol Neurosurg Psychiatry 2008;79:289-93.
11. McKhann GM, Cornblath DR, Griffin JW, Ho TW, Li CY, Jiang Z, *et al.* Acute motor axonal neuropathy: A frequent cause of acute flaccid paralysis in China. Ann Neurol 1993;33:333-42.
12. Lawn ND, Wijdicks EF. Fatal Guillain-Barré syndrome. Neurology 1999;52:635-8.
13. Chiba A, Kusunoki S, Obata H, Machinami R, Kanazawa I. Serum anti-GQ1b IgG antibody is associated with ophthalmoplegia in miller fisher syndrome and Guillain-Barré syndrome: Clinical and immunohistochemical studies. Neurology 1993;43:1911-7.

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